IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF TEXAS
MARSHALL DIVISION

CENTOCOR ORTHO BIOTECH, INC. and NEW YORK UNIVERSITY,	)	
Plaintiffs,	)	**CIVIL ACTION NO. 2:07CV139**
v.	)	**JURY TRIAL DEMANDED**
ABBOTT LABORATORIES, ABBOTT BIORESEARCH CENTER, INC., and ABBOTT BIOTECHNOLOGY LTD.	)	
Defendants.	)	

PLAINTIFFS CENTOCOR ORTHO BIOTECH, INC.AND NEW YORK UNIVERSITY'S PROPOSED FINDINGS OF FACT AND CONCLUSIONS OF LAW ON INEQUITABLE CONDUCT, PROSECUTION LACHES, AND INDEFINITENESS

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Plaintiffs Centocor Ortho Biotech, Inc. and New York University (collectively, "Centocor") submit the following proposed findings of fact and conclusions of law on the issues of inequitable conduct, prosecution laches, and indefiniteness.[1]

PROPOSED FINDINGS OF FACT

I. THE PARTIES AND BACKGROUND OF THIS LAWSUIT

1. Plaintiff Centocor Ortho Biotech, Inc. ("Centocor") is a corporation organized under the laws of the Commonwealth of Pennsylvania and maintains its principal place of business at 800/850 Ridgeview Drive, Horsham, Pennsylvania 19044 (D.I. 228 at 5).

2. Plaintiff New York University ("NYU") is a research university organized as a corporation and existing under the laws of the State of New York and having a place of business at 70 Washington Square South, New York, New York 10012 (D.I. 228 at 6).

3. Defendant Abbott Laboratories ("Abbott Labs") is a corporation organized and existing under the laws of the State of Illinois with a place of business as 100 Abbott Park Road, Abbott Park, Illinois 60064 (D.I. 228 at 6).

4. Defendant Abbott Bioresearch Center ("ABC") is a wholly owned subsidiary corporation of Abbott Labs that is organized under the laws of the State of Delaware with a place of business at 100 Research Drive, Worchester, Massachusetts 01605 (D.I. 228 at 6).

5. Defendant Abbott Biotechnology Ltd. ("ABL") is a wholly owned subsidiary corporation of Abbott Labs that is organized under the laws of Bermuda with a place of business at Carr #2 Km. 59.2, Segundo Piso, Barceloneta, Puerto Rico 00617 (D.I. 228 at 6).

6. In its Infringement Contentions dated July 10, 2008, Centocor alleged that the Abbott defendants infringe claims 1, 2, 3, 8, 9, 11, 13, 14, 15, and 20 of U.S. Patent No.

[1] To the extent that any proposed finding of fact is deemed to be an issue of law, Centocor requests that it be so considered. To the extent that any proposed conclusion of law is deemed to be an issue of fact, Centocor requests that this also be so considered.

7,070,775 (the "775 Patent") and claims 3, 9, 10, 11, 12, and 14 of U.S. Patent No. 7,276,239 Patent (the "239 Patent") (Ex. 1 at 2). (Hereinafter, the Abbott entities may be collectively referred to as "Abbott".)

7. Centocor, Inc. changed its name to a new corporate name, Centocor Ortho Biotech, Inc. (D.I. 228 at 6).

8. Centocor Ortho Biotech and NYU are co-assignees of the 775 Patent entitled "Recombinant A2-Specific TNFα Specific Antibodies" (D.I. 228 at 6 & PX 1). The 775 Patent was issued by the United States Patent and Trademark Office ("USPTO") on July 4, 2006 (PX 1).

9. Centocor Ortho Biotech and NYU are co-assignees of the 239 Patent entitled "Recombinant A2-Specific TNFα Specific Antibodies" (D.I. 228 at 6 & PX 2). The 239 Patent was issued by the USPTO on October 2, 2007 (PX 2). Hereinafter, Centocor Ortho Biotech and NYU may be referred to collectively as "Centocor."

10. On April 16, 2007, Centocor filed suit against Abbott Labs, alleging that Abbott Labs infringes the 775 Patent (D.I. 1). On March 18, 2008, Centocor amended its Complaint to add the allegation that Abbott Labs also infringes the 239 Patent (D.I. 56). On June 19, 2008, Centocor amended its Complaint again to add Abbott Bioresearch Center and Abbott Biotechnology Ltd. as defendants to this suit (D.I. 70).

11. The jury and non-jury issues were bifurcated for trial (D.I. 211). Based on that Bifurcation Order, the parties agreed that the issues of indefiniteness, prosecution laches, and inequitable conduct would not be presented to the jury (D.I. 228 at 8).

12. Before the jury trial, based on certain stipulations, the asserted claims were narrowed to claims 2, 3, 14, and 15 of the 775 Patent (D.I. 241). The jury trial commenced on

June 22, 2009. In that trial, Centocor asserted that Abbott's Humira product infringes each of the asserted claims, that Abbott's infringement is and has been willful, and that Centocor was entitled to lost profits and reasonable royalty damages on account of Abbott's infringement. Abbott asserted that it did not infringe any of the asserted claims, and that the asserted claims were invalid for anticipation, lack of written description, and lack of enablement.

13. On June 29, 2009, the jury returned a verdict finding that Abbott infringes claims 2, 3, 14, and 15 of the 775 Patent, that Abbott's infringement is and has been willful, that the asserted claims are not invalid for anticipation, lack of written description, and lack of enablement, and that Centocor was entitled to both lost profit and reasonable royalty damages on account of Abbott's infringement (D.I. 261).

14. The Court conducted a bench trial on August 4, 2009, to consider Abbott's claims of indefiniteness, prosecution laches, and inequitable conduct. The Court has considered the live testimony, testimony by deposition designations, documentary evidence as well as arguments presented on these issues, and makes the additional findings of fact set forth below.

II. THE 775 AND 239 PATENTS

A. Prosecution Of The Applications That Issued As The 775 And 239 Patents

15. The application that issued as the 775 Patent was filed on July 18, 2002, and was assigned to Examiner Phillip Gambel. Among the references cited to the Examiner in an information disclosure statement submitted on September 23, 2002 was a 1990 paper authored by Achim Moller (the "Moller paper") (Ex. 2 at CCOR00000337 and PX 192). The examiner indicated that he considered the Moller paper by initialing the information disclosure statement (*id*. at CCOR00000439). The Moller paper was also cited twice in the specification of the 775 Patent- once in the background section and once in the section of the specification describing the

species specificity profile for the A2 antibody (*id*. at CCOR00000174 and CCOOR00000251-52).

16. It is clear from Examiner Gambel's search notes that he reviewed the patents and applications in the Le cA2 TNF family (*id*. at CCOR00000454). It is also clear that he reviewed the parent patents for priority (*id*. at CCOR00000511).

17. On December 28, 2005, Examiner Gambel allowed the application that issued as the 775 Patent on July 4, 2006 (*id*. at CCOR00000507-510).

18. The Court finds that there is every indication from the prosecution history for the 775 Patent that the application on which it issued was given careful consideration by the Patent Examiner, that the Examiner had a duty to be, and was, aware of the prosecution of prior applications in the family, and the Examiner considered, among other things, the Moller paper prior to allowing the 775 Patent claims.

19. The application that issued as the 239 Patent was filed on December 20, 2005, and was assigned to Examiner Phillip Gambel. Among the references cited to the Examiner in an information disclosure statement submitted on February 8, 2006 was the Moller paper (Ex. 3 at CCOR00020543). The examiner indicated that he considered the Moller paper by initialing the information disclosure statement (*id*. at CCOR00021037). The Moller paper was also cited twice in the specification of the 239 Patent- once in the background section and once in the section of the specification describing the species specificity profile for the A2 antibody (*id*. at CCOR00019969 and CCOOR00020046).

20. It is clear from Examiner Gambel's search notes that he reviewed the patents and applications in the related patents and applications in the family (*id*. at CCOR00021052).

21. On June 26, 2007, Examiner Gambel allowed the application that issued as the 239 Patent on October 2, 2007 (*id*. at CCOR00021018-023).

22. The Court finds that there is every indication from the prosecution history for the 239 Patent that the application on which it issued was given careful consideration by the Patent Examiner, that the Examiner had a duty to be, and was, aware of the prosecution of prior applications in the family, and the Examiner considered, among other things, the Moller paper prior to allowing the 239 Patent claims.

23. Abbott has not identified any statement or argument made during prosecution of the 775 and/or 239 Patents that Abbott alleges to be false or misleading. Accordingly, the Court finds that there were no false or misleading statements or arguments made during the prosecution of applications that issued as the 775 and/or 239 Patents.

24. The only inequitable conduct allegation made by Abbott that is directed to the prosecution of the applications that issued as the 775 and 239 Patents (rather than to the prosecution of other applications in the same patent family) is Abbott's allegation that Centocor failed to cite to the Examiner particular rejections from certain prior applications relating to the Moller paper. The Court finds, however, that the Examiner who examined the applications that issued as the 775 and 239 Patents had a duty to be, and was, aware of the history of prosecution of prior applications in the 775 and 239 Patent family, and also that the Examiner considered, among other things, the Moller paper prior to allowing the 775 and 239 Patent claims.

25. Also, although Abbott had a patent attorney expert testify at trial, that expert did not indicate that, as a matter of good or required practice, Centocor should have summarized the history of prosecution and rejections of applications in the same family. The Court does not find

any basis for charging Centocor with a duty of summarizing the history of prior prosecution events in the direct family.

26. Abbott has also failed to come forward with any evidence of intent to deceive the patent office during prosecution of the applications that issued as the 775 and 239 Patents. Abbott deposed the attorney who prosecuted those patent applications, Deirdre Sanders, and introduced portions of her deposition testimony at trial. But Abbott has not pointed to a scintilla of evidence that she – or anyone else involved in prosecution of the applications that issued as the 775 and 239 Patents – ever had any intent to deceive or mislead the Patent Office.

27. Accordingly, the Court finds that Abbott has not met its burden of proving by clear and convincing evidence that there was any inequitable conduct during prosecution of the applications that issued as the 775 and/or 239 Patents.

B. Prosecution Of Earlier Applications In The Family

28. Abbott alleges that certain arguments made during prosecution of certain applications in the same family as the 775 and 239 Patents were false and/or misleading.

29. The application that issued as the 775 Patent was filed on July 18, 2002, as a continuation of application No. 09/756,398, filed on Jan. 8, 2001, now U.S. Patent No. 6,835,823, which is a division of application No. 09/133,119 filed on Aug. 12, 1998, now U.S. Patent No. 6,277,969, which is a division of application No. 08/570,674, filed on Dec. 11, 1995, now abandoned, which is a continuation-in-part of application No. 08/324,799, filed on Oct. 18, 1994, now U.S. Patent No. 5,698,195, which is a continuation-in-part of application No. 08/192,102 (the "102 application"), filed on Feb. 4, 1994, now U.S. Patent No. 5,656,272, and a continuation-in-part of application No. 08/192,861 (the "861 application"), also filed on Feb. 4, 1994, now U.S. Patent No. 5,919,452, and a continuation-in-part of application No. 08/192,093 ("the 093 application"), also filed on Feb. 4, 1994, now U.S. Patent No. 6,284,471, which is a

continuation-in-part of application No. 08/010,406 filed on Jan. 29, 1993, now abandoned, which is a continuation-in-part of application No. 07/943,852, filed on Sept. 11, 1992, now abandoned, which is a continuation-in-part of application No. 07/853,606, filed on Mar. 18, 1992, now abandoned, which is a continuation-in-part of application No. 07/670,827, filed on Mar. 18, 1991, now abandoned (PX 1).

30. The 239 Patent application was filed on December 20, 2005 as a division of the application that issued as the 775 patent (PX 2).

31. Abbott alleges that certain arguments made during prosecution of each of the three applications filed on February 4, 1994 (the 102, 861, and 093 applications or collectively "the 1994 applications") were false or misleading.

32. The specific arguments identified by Abbott were made in response to Examiner rejections based on the Moller and Rathjen references, although Abbott only contends that the arguments were false and/or misleading with respect to the disclosure of the Moller paper.

33. The rejections in each of the three pending applications were nearly identical and stated that:

> The Moller et al. references teach monoclonal antibody M195 which appears to be the same as the antibody of the present invention. M195 is functionally similar to the A2 antibody as characterized in the specification, in exhibiting high affinity binding to TNF-alpha, neutralizing TNF-alpha but not TNF beta (see p. 164 Table 2) binding to human and chimpanzee TNF but not TNF from baboon, rhesus monkey or cynomolgus monkey (e.g. Cytokine, p. 164 col. 1). In view of those similarities, the A2 and M195 antibodies appear to have the same or similar epitope binding specificities and M195 is expected to have the properties recited in the instant claims.

(Ex. 4 at ABT01363639; Ex. 5 at ABT01365150; Ex. 6 at ABT01364594).

34. The claims pending in the 102 and 861 applications at the relevant time were directed to methods of treating Crohn's disease by administering effective amounts of chimeric anti-TNFα antibodies. Claim 106 of the 861 application is representative and read as follows:

> 106. A method for treating Crohn's disease in a human comprising administering to the human an effective TNF-inhibiting amount of an anti-TNF chimeric antibody, wherein said anti-TNF chimeric antibody comprises a non-human variable region or a TNF-binding portion thereof and a human constant region.

(Ex. 5 at ABT01365111).

35. The claims pending in the 093 application at the relevant time were directed to chimeric anti-TNFα antibodies. Claim 1 is representative and read as follows:

> 1. A chimeric antibody, comprising at least part of a human immunoglobulin constant region and at least part of a non-human immunoglobulin variable region, said antibody capable of binding an epitope specific for human tumor necrosis factor TNF**.**

(Ex. 4 at ABT01363162).

36. In response to the rejection of the then-pending claims over the Moller paper, the applicants, through their attorney Ms. Carolyn Elmore, responded by arguing:

> Moller [does] not explicitly describe the epitopic specificity of the monoclonal antibodies. Thus, any rejection or assertion that the present claims describing antibodies of the preferred epitopic specificity are unpatentable over [this reference] requires the assumption that the antibodies inherently inhibit A2 binding to TNF.
>
> Inherency need to be a definite result not a mere possibility, There is nothing cited in the prior art which would suggest that antibodies which recognize the A2 epitope would neutralize TNFα. Far from being inherent, *it is unlikely that any of the antibodies described in the foregoing studies are specific for the same TNFα neutralizing epitope(s) as Applicants' claimed monoclonal antibodies*. The cited references do not describe *in vivo* neutralizing ability of any antibody described for use in the treatment of human, nor do they teach the specific epitope to which the preferred antibodies of the claims bind. TNFα is known to contain many epitopes. *A skilled artisan, on the basis of the information disclosed in these references, would not conclude that any of the prior art antibodies are identical to or contain the features of the antibodies claimed by the Applicants*.
>
> In distinction, the antibodies prepared herein have been demonstrated to be capable of neutralizing TNFα in a clinical setting with superior results.

(Ex. 4 at ABT01363659; *see* Ex. 5 at ABT01365185; *see* Ex. 6 at ABT01364629). The portions of this response that Abbott has alleged to be false or misleading are emphasized for purposes of identification and will be discussed in turn below.

1. Argument 1: "It Is Unlikely That Any Of The Antibodies Described In The Foregoing Studies Are Specific For The Same TNFα Neutralizing Epitope(s) As Applicants' Claimed Monoclonal Antibodies"

37. Abbott has specifically identified the argument made by Attorney Elmore that "it is unlikely that any of the antibodies described in the foregoing studies are specific for the same TNFα neutralizing epitope(s) as Applicants' claimed monoclonal antibodies" and has alleged that this argument is false or misleading. The Court finds, however, that the argument is true. Not only is the Moller antibody unlikely to bind to the same epitope as A2 and/or cA2, the evidence shows that in fact it **does not** bind to the same epitope as A2 and/or cA2.

a) MAK-195 and A2 Do Not Bind To The Same Epitope On TNFα

38. As defined in the relevant patent specifications, "[t]he term 'epitope' is meant to refer to that portion of any molecule capable of being recognized by and bound by an antibody at one or more of the [antibody's] antigen binding regions. Epitopes usually consist of chemically active surface groupings of molecules such as amino acids or sugar side chains and have specific three dimensional structural characteristics as well as specific charge characteristics." (PX 1 at 13:15-21).

39. According to Abbott scientist, Dr. Salfeld, in order to determine whether MAK-195 and A2 bind to the same, or even to a similar, epitope on TNF, scientists would need to solve the x-ray crystal structure of A2 with human TNF and the x-ray crystal structure of MAK-195 with human TNF (Salfeld, Bench Trial at 199:12-20) (Ex. 7).

40. Centocor has not had access to the x-ray crystal structure of MAK-195 bound to TNFα, but Abbott generated this structure in 2009 and has kept it confidential since then (Marks, Bench Trial at 87:15 – 88:17 (Ex. 7); Argiriadi Dep. at 190:1-9, 191:5-9 (Ex. 8)). TNF is a trimer, meaning that it is made up of three identical strands of amino acids (Marks, Bench Trial at 89:12-14) (Ex. 7). The MAK-195 crystal structure generated by Abbott in 2009 shows that MAK-195 binds to a single strand or single monomer of the TNF molecule (*id*. at 89:15-20).

41. There is no crystal structure available at all for A2 or cA2 bound to TNF (*id*. at 88:18-21). But, for purposes of this case, Dr. Marks modeled the binding site for the cA2-TNF alpha complex based on information in a technical report generated by Centocor (*id*. at 88:22 – 89:7, referring to DX 412 (Ex. 9)).

42. That Centocor report reveals that the cA2 epitope (which is identical to the A2 epitope) (Marks, Bench Trial at 109:11-16) (Ex. 7) is significantly different from the MAK-195 epitope. In contrast to the MAK-195 epitope that is found on a single monomer of the TNF trimer, the "sequences [of the cA2/A2 epitope] form a loose cluster in the region between two monomer units" (DX 412 at CCOR00021290). "Importantly, sequences 25-31, 70-82 and 138 belong to one monomer unit while sequences 113-119 and 145-147 are on an adjacent unit" (*id*.). In other words, the cA2/A2 epitope "span[s] two chains" (*id*. at CCOR00021296; *see also id*. at CCOR00021302 ("[T]he monomer units [of TNF] contain only part of the intact epitope")).

43. Consistent with the argument made to the Patent Office by Attorney Elmore, Dr. Marks also admitted on cross-examination that the epitopes for cA2/A2 and MAK-195 are different. When asked to compare his model of the cA2/A2 epitope to the MAK-195 epitope (determined from the Abbott crystal structure), Dr. Marks was forced to admit that "it would

appear that there are differences between the epitopes" (Marks, Bench Trial at 93:24-94:4) (Ex. 7).

44. There is additional evidence that supports the conclusion that the A2/cA2 epitope and the MAK-195 epitope are not the same. Abbott scientist Paul Sakorafas studied a binding fragment of MAK-195 and cA2 side-by-side in "pair-wise epitope mapping" experiments (DX 440 at ABT00161673). The two antibodies reacted differently in the tests – some proteins competed with cA2 but not with MAK-195 (*id*.). According to Dr. Salfeld, that result "certainly gives you pause to see a difference" (Salfeld Dep. at 209:16 – 210:14) (Ex. 9).

45. Dr. Marks agreed that one explanation of the Sakorafas data is that there is a difference in the binding sites of the MAK-195 fragment and cA2 (Marks, Bench Trial at 100:11-16) (Ex. 7). Indeed, in support of his non-infringement arguments, Dr. Marks interpreted similar Abbott results with Humira and cA2 as showing a "significant difference" in the epitopes (*id*. at 101:5-18).

46. Accordingly, the Court finds that Abbott has not met its burden of proving by clear and convincing evidence that the argument made by Attorney Elmore that "it is unlikely that any of the antibodies described in the foregoing studies [including MAK-195] are specific for the same TNFα neutralizing epitope(s) as Applicants' claimed monoclonal antibodies" is false and/or misleading. Indeed, as Abbott's expert admitted, these antibodies *do not* bind to the same epitope on TNFα.

b) Shared Species Specificity Does Not Indicate That Two Antibodies Bind To The Same Epitope On TNFα

47. Abbott has further alleged that Centocor should have known that the argument made by Attorney Elmore that "it is unlikely that any of the antibodies described in the foregoing studies are specific for the same TNFα neutralizing epitope(s) as Applicants' claimed

monoclonal antibodies" was false or misleading because A2 and MAK-195 share species specificity. As set forth above, the Court finds, however, that the argument made by Attorney Elmore is true. Not only is the Moller antibody unlikely to bind to the same epitope as A2 and/or cA2, Abbott's expert admitted that it does not bind to the same epitope as A2 and/or cA2. Moreover, the Court finds that shared species specificity does not indicate that two antibodies bind to the same epitope.

48. Scientists from Centocor, NYU, and Abbott agree that the results of species specificity experiments for two antibodies do not provide enough information to know whether those antibodies bind to the same epitope. Dr. David Knight from Centocor testified that the "species cross-reactivity profile does not give you full information about an epitope" (Knight, Bench Trial at 203:20-204:4) (Ex. 7). Dr. Vilcek from NYU agreed that the species specificity is "by itself not sufficient" to determine whether two antibodies bind to the same epitope and testified that he had no direct evidence that MAK-195 and A2 would recognize the same epitope (Vilcek, Bench Trial at 201:12 – 202:1) (Ex. 7). According to Abbott scientist Dr. Salfeld, in order to determine whether MAK-195 and A2 bind to the same, or even to a similar, epitope on TNF, scientists would need to solve the x-ray crystal structure of A2 with human TNF and the x-ray crystal structure of MAK-195 with human TNF (Salfeld, Bench Trial at 199:12-20) (Ex. 7).

49. The specification of the 775 Patent also does not support Abbott's argument that the species specificity profiles of A2 and cA2 indicate that the two antibodies must bind to the same epitope. Dr. Marks answered a question in his direct examination that made it seem like a portion of the patent states that A2 and cA2 are expected to bind to the same epitope because of their species specificity profiles (Marks, Bench Trial at 58:5-10) (Ex. 7). But as Dr. Marks admitted on cross-examination, the reason that A2 and cA2 are expected to have identical

epitopes is that they share identical variable regions (*id*. at 109:11-18). Furthermore, the passage in the specification that refers to the A2 and cA2 epitopes says absolutely nothing about species specificity (*id*. at 109:23 – 110:8, referring to PX 1 at 49:1-4). Thus, the species specificity profiles do not themselves show that the epitopes are the same.

50. The crux of Abbott's argument on the shared species specificity seems to be that the fact that human and baboon TNF differ in only one amino acid (residue 138), coupled with the fact that MAK-195 and A2 bind to human but not baboon TNF, means that their binding sites must be the same. But the Court does not find support for that assertion. To the contrary, Abbott's expert, Dr. Marks, admitted that, even though residue 138 is implicated in the binding of both MAK-195 and cA2, the two antibodies do not bind to the same epitope (Marks, Bench Trial at 95:2-15) (Ex. 7). Thus, contrary to Abbott's argument, the Court finds that the fact that residue 138 is implicated in binding of both antibodies does not mean that their epitopes are the same.

c) Neutralizing Ability Does Not Indicate That Two Antibodies Bind To The Same Epitope On TNFα

51. Abbott has further alleged that Centocor should have known that the argument made by Attorney Elmore that "it is unlikely that any of the antibodies described in the foregoing studies are specific for the same TNFα neutralizing epitope(s) as Applicants' claimed monoclonal antibodies" was false or misleading because A2 and MAK-195 both neutralize TNFα. As set forth above, the Court finds, however, that the argument made by Attorney Elmore is true. Not only is the Moller antibody unlikely to bind to the same epitope as A2 and/or cA2, Abbott's expert admitted that it does not bind to the same epitope as A2 and/or cA2. Moreover, the Court finds that neutralizing ability does not indicate that two antibodies bind to the same epitope.

52. For anti-TNFα antibodies in particular, scientists know that different antibodies to the antigen (human TNFα) can bind at different epitopes (Marks, Bench Trial at 105:14-22 (Ex. 7); PX 195 at 33). Indeed, different neutralizing antibodies to TNF are known to bind to different epitopes (Marks, Bench Trial at 105:14-22 (Ex. 7); PX 195 at 22, 33). This is explicitly disclosed in the Moller paper relied on by Abbott; different "neutralizing monoclonal anti-TNFα antibodies … bind to distinct antigenic epitopes" (PX 192 at CCOR00011488; Marks, Bench Trial at 106:5-14 (Ex. 7)).

53. The Rathjen reference that was also the subject of the Examiner rejections, indicates that there are a number of different anti-TNF antibodies that have neutralizing activity but bind to different epitopes on TNF alpha (Marks, Bench Trial at 105:14–22) (Ex. 7). The Court, accordingly, finds that the fact that two antibodies are neutralizing does not indicate that they bind to the same epitope.

d) "Same Or Similar" Is Different Than "The Same"

54. Abbott further contends that the argument made by Attorney Elmore that "it is unlikely that any of the antibodies described in the foregoing studies are specific for the same TNFα neutralizing epitope(s) as Applicants' claimed monoclonal antibodies" should be interpreted as stating something beyond the actual words. To this end, Abbott argues that "the same" should be construed to mean "the same or similar" because in other places in the prosecution history where Attorney Elmore is talking about competition with A2, she refers to antibodies having the same or a similar epitope.

55. The Court rejects this argument as baseless. In the first instance, the fact that Attorney Elmore said "the same or similar" in certain circumstances when referring to competition but used the phrase "the same" in the specific argument at issue, means that she drew a distinction between the two. Further, Abbott has not presented any persuasive argument

or evidence as to why two independent arguments made during prosecution should be selectively juxtaposed in order to alter the plain meaning of one of the arguments.

56. There is a difference between binding to the same epitope and binding to the same or a similar epitope and that difference is recognized in this field (Marks, Bench Trial at 85:18-21) (Ex. 7). Indeed, at trial, Abbott's expert, Dr. Marks, admitted that someone in this field would not consider the phrase "the same epitope" to encompass a "the same or a similar epitope" (*id*. at 84:12-17).

57. Abbott's expert, Dr. Marks, further admitted at trial that he had previously taken the position in this litigation that "[i]f two antibodies do not bind to the same epitope, they interact in entirely different ways with the antigen and consequently with each other" (*id*. at 86:21 – 87:2, 114:4-21). Thus, to the extent Dr. Marks tried to suggest that saying the "same epitope" is somehow synonymous with saying a "similar epitope," that suggestion is not credible. The Court finds that binding to the "same" epitope means something different than binding to a "similar" epitope, and that there is no basis for construing Attorney Elmore's argument to mean anything other than what it says.

58. Moreover, to this day, even with x-ray crystal structure information available for MAK-195, no one at Abbott has ever recognized that MAK-195 and A2 would compete for binding to TNFα (Abbott Response to Interrogatory No. 30) (Ex. 10). So even if the argument made by Attorney Elmore could be contorted to somehow suggest that she was saying that MAK-195 and A2 do not compete for binding (as Abbott now suggests), there is no evidence proving that such an argument would be false or misleading. Abbott's expert, Dr. Marks, admitted as much at trial in testifying that the fact that two antibodies compete, does not, in any event, mean that they share the same epitope (Marks, Bench Trial at 102:25 – 103:9) (Ex. 7).

59. The Court deems the carefully-worded testimony of Dr. Marks that it was "more likely than unlikely" that the A2 and MAK-195 epitopes are the same (*id*. at 79:18-25, 82:14-24) to be unpersuasive. That testimony is directly contradicted by the Centocor technical report, the Sakorafas epitope mapping studies, and Dr. Marks's own models of the epitopes and admissions on cross-examination.

60. The Court further finds that Abbott has failed to meet its burden of proving by clear and convincing evidence that the argument made by Attorney Elmore that "it is unlikely that any of the antibodies described in the foregoing studies are specific for the same TNFα neutralizing epitope(s) as Applicants' claimed monoclonal antibodies" is somehow false or misleading.

2. Argument 2: "A Skilled Artisan, On The Basis Of The Information Disclosed In These References, Would Not Conclude That Any Of The Prior Art Antibodies Are Identical To Or Contain The Features Of The Antibodies Claimed By The Applicants"

61. Abbott has also specifically identified the argument made by Attorney Elmore that "[a] skilled artisan, on the basis of the information disclosed in these references, would not conclude that any of the prior art antibodies are identical to or contain the features of the antibodies claimed by the Applicants," and has alleged that this argument is false or misleading. The Court finds, however, that the argument is true.

62. Abbott does not contend that MAK-195 and the "antibodies claimed" in the 1994 applications are identical, so the truth of this argument is undisputed. It is also undisputed that Moller does not describe the features of the claimed antibodies, including preparation of chimeric anti-TNFα antibodies, nor does Moller provide a reasonable expectation of achieving an antibody of reduced immunogenicity and/or possessing a therapeutic benefit, or suggest unexpected results in a clinical setting. The Court, accordingly, finds that the MAK-195

antibody is not identical to and does not contain the features of the antibodies claimed in the 1994 applications.

63. Abbott's contentions on this issue are rather circumscribed. In order to contend that the argument made by Attorney Elmore is somehow false or misleading, Abbott points to characteristics of the MAK-195 antibody that were not the subject of Attorney Elmore's argument about the features of the claimed antibodies. Specifically, Abbott points to the affinity, neutralizing ability, and species specificity of MAK-195 (Marks, Bench Trial at 71:24 – 72:5) (Ex. 7). But Abbott has not correlated these characteristics with the "features of the antibodies claimed" – *i.e.*, the features of the antibodies that were then the subject of claims pending in the 1994 applications and the subject of Attorney Elmore's arguments.

64. The context of Attorney Elmore's argument makes clear that the "features of the antibodies claimed" does not refer to the affinity, neutralization activity, or species specificity characteristics. Indeed, most of the pending claims did not even refer to such characteristics in the claimed chimeric anti-TNFα antibodies and methods for treating Crohn's disease. Furthermore, the species specificity and in vitro neutralizing activity of MAK-195 were expressly referred to in the patent applications (*e.g.*, PX 1 at 2:61-3:1, 49:42-46, 49:60-61; *see infra* Proposed Finding of Facts ¶¶ 87-88), and those properties, as well as the high affinity, were therefore known to the examiner and cited in the pending rejections (*see supra* Proposed Finding of Fact ¶ 33). Rather than referring to those properties, Attorney Elmore's argument, in context, was as follows:

> A skilled artisan, on the basis of the information disclosed in these references, would not conclude that any of the prior art antibodies are identical to or contain the features of the antibodies claimed by the Applicants.
>
> In distinction, the antibodies prepared herein have been demonstrated to be capable of neutralizing TNFα in a clinical setting with superior results.

> Thus, the [Moller paper does] not describe or suggest the preparation of chimeric antibodies which bind TNFα, [does] not provide a reasonable expectation of achieving an antibody of reduced immunogenicity and/or possessing a therapeutic benefit and [does] not reasonably suggest the unexpected and superior results achieved and described herein were possible.

(Ex. 4 at ABT01363659-60).

65. As set forth above, the distinguishing features of the claimed antibodies that were the subject of Attorney Elmore's argument were the ability to neutralize TNFα in a clinical setting, the preparation of chimeric antibodies that bind TNFα, the reduced immunogenicity and/or therapeutic benefit, and the unexpected and superior results. Importantly, it is undisputed that MAK-195 does not contain the features to which Attorney Elmore was referring (Marks, Bench Trial at 108:8 – 109:1) (Ex. 7). It is therefore undisputed that MAK-195 does not contain the features of the antibodies claimed by the applicants.

66. Attorney Elmore also expressly pointed out to the patent examiner on multiple occasions that the Moller paper describes a "species specific murine monoclonal antibody, mAb 195, which neutralizes human TNF" (Ex. 4 at ABT01363698; Ex. 5 at ABT01365265-66; Ex. 6 at ABT01364628). Attorney Elmore distinguished Moller because it did not, for example, describe the desirability of using IgG1 antibodies (Ex. 4 at ABT01363701-02; Ex. 5 at ABT01365270-71) or "suggest the treatment of humans suffering from Crohn's disease with a chimeric antibody" (Ex. 6 at ABT01364628). It is also undisputed that those features, which were in the pending claims, are not found in MAK-195 (Marks, Bench Trial at 108:13 – 109:5) (Ex. 7).

67. The Court finds that Abbott has therefore failed to prove by clear and convincing evidence that the argument about antibody features was untrue.

3. There Is No Evidence Of Deceptive Intent

68. Abbott has not identified any evidence that the inventors, attorney, or anyone else involved in the prosecution of the 1994 applications had any intent to deceive the PTO. Instead, Abbott has asked the Court to infer such an intent. The Court finds, however, that the evidence does not support any such inference.

a) Deceptive Intent Cannot Be Inferred From The Vilcek Letters

69. Abbott's primary argument for an inference of intent is based on a few letters drafted by one of the inventors in 1990. The Court does not find that these letters provide clear and convincing evidence of deceptive intent.

70. On August 1, 1990 (DX 55), Dr. Jan Vilcek, one of the NYU inventors, wrote to his co-inventor, Dr. Scott Siegel at Centocor, informing him of a paper authored by Dr. Achim Moller and others, titled "Monoclonal Antibodies to Human Tumor Necrosis Factor-α: In Vitro and In Vivo Application," Cytokine, 2(3):162-169 (1990) (PX 192).

71. Dr. Vilcek informed Dr. Siegel that the Moller paper reports on two monoclonal antibodies to human TNF-α, one of which neutralized human and chimpanzee TNF-α, but not TNF-α from other primate species (DX 55). The mouse monoclonal anti-TNF-α antibody designated A2, had also been tested against baboon TNF-α and failed to neutralize baboon TNF-α, and Dr. Vilcek reported that A2 would be tested to determine whether it neutralized chimpanzee TNF (*id*.).

72. Just over two weeks later, on August 15, 1990, Dr. Vilcek sent a second letter to Dr. Siegel in which he reported that the A2 did neutralize chimpanzee TNF-α (DX 56). He recognized that, in this respect, A2 "resembled" Moller's MAK-195 antibody (*id*.).

73. Abbott contends that this statement by Dr. Vilcek, recognizing that A2 resembled MAK-195 in its species specificity, is evidence that the argument made by Attorney Elmore that "it is unlikely that any of the antibodies described in the foregoing studies are specific for the same TNFα neutralizing epitope(s) as Applicants' claimed monoclonal antibodies" was made with the intent to deceive the PTO. But the Court finds that the evidence does not support this contention.

74. First, as set forth above, the Court finds, not only that the Moller antibody is unlikely to bind to the same epitope as A2 and/or cA2, but, moreover, as Abbott's expert admitted, it does not bind to the same epitope as A2 and/or cA2. The Court cannot infer an intent to deceive the PTO based on a true argument.

75. Moreover, as set forth above, the Court finds that shared species specificity does not indicate that two antibodies bind to the same epitope. This was confirmed by scientists from Centocor and Abbott (Knight, Bench Trial at 203:20-204:4; Salfeld, Bench Trial at 199:12-20) (Ex. 7), and confirmed by Dr. Vilcek himself (Vilcek, Bench Trial at 201:12 – 202:1) (Ex. 7). Indeed, the crux of Abbott's argument was undermined by its own expert, Dr. Marks, who admitted that, even though residue 138 (the only residue that is different in human and baboon TNF) is implicated in the binding of both MAK-195 and cA2, the epitopes for these two antibodies are not the same (Marks, Bench Trial at 95:2-15) (Ex. 7).

76. Contrary to Abbott's allegations, a Progress Report for 1993 to 1994 also does not show that Dr. Vilcek withheld any information about MAK-195. That report describes studies on cA2 requested by the FDA to "determine whether toxicology/safety studies in animal models would be warranted" (DX 113 at NYU00002606). In the context of looking for animal models for cA2 testing, the report states that the species specificity of *cA2* is "quite startling" and that

"baboon TNF is known to differ from human TNF-α in a single amino acid" (*id*. at NYU00002608). But, the report does not refer to the cA2 epitope or to MAK-195 at all. And, contrary to Abbott's assertions, the one amino acid difference does not show that MAK-195 and cA2 have the same or similar epitopes. To the contrary, Abbott's expert, Dr. Marks, admitted that, based on his own models of the MAK-195 and cA2 epitopes, the two antibodies do not bind to the same epitope.

77. The Court further finds that, to the extent an inference can be drawn in this context, it must be inferred that the inventors interest in the shared species specificity was based on an interest in developing a model for FDA purposes. Specifically, in the August 1, 1990 letter, Dr. Vilcek specifically mentioned that "the apparent lack of neutralizing activity of MAb A2 against primate TNF preparations will ultimately work to our advantage, as we may be able to convince the FDA that there is no animal model available for the evaluation of" A2 (DX 55). Centocor inventor Scott Siegel also described the difficulties in testing cA2 in animals, because the species specificity meant that chimpanzee was the only "appropriate" animal model (DX 29 at CCOR00012544). Dr. Siegel mentioned the Moller paper only because it reported the testing of antibodies on mice that had been injected with human TNF, but he wrote that the relevance of that testing was "obviously subject to question, since this may differ greatly from what occurs as a response to disease" (*id*. at CCOR00012545). Abbott's expert Dr. Marks agreed that species specificity results are useful for identifying animals to use in preclinical models and for developing clinical trials (Marks, Bench Trial at 110:13 – 111:1) (Ex. 7). Thus, it is reasonable to infer that the inventors were interested in the species specificity of cA2 because of their efforts to identify an animal model for testing the antibody.

78. Further, it is not enough for Abbott to generally allege that someone had the intent to deceive the PTO. Even if the Vilcek letters could be construed as somehow inconsistent with arguments made to the PTO by Attorney Elmore, Abbott has provided no evidence that she ever saw the letters from Dr. Vilcek that Abbott now relies on, or that Dr. Vilcek ever saw the arguments made by Attorney Elmore to the PTO. Because those letters were written five years before Attorney Elmore made her arguments, it is unlikely that she was aware of them. The Court finds that it is not reasonable to infer from the evidence that either Attorney Elmore or the inventors had an intent to deceive the Patent Office.

79. Accordingly, the Court does not find that Abbott has provided clear and convincing evidence from which an intent to deceive the PTO can be inferred.

b) Deceptive Intent Cannot Be Inferred Based On A Lack Of Evidence That Abbott Failed To Pursue

80. Abbott's second argument for an inference of intent is based on a lack of explanation from Attorney Elmore herself that the subject arguments were made without an intent to deceive the PTO. The Court does not find this to be a basis for finding deceptive intent, particularly since Abbott failed to pursue the relevant discovery.

81. The prosecution history clearly identifies Attorney Elmore as the author of the subject arguments, but Abbott chose not to depose Ms. Elmore to find out whether she was aware of the Vilcek letters or not, or whether she has any other information relevant to the issue of intent.

82. Abbott chose instead to take a 30(b)(6) deposition on the broad topic of "[t]he prosecution of the applications leading to the Centocor Patents and of any patents or patent applications to which the Centocor Patents claim priority." Centocor objected to that topic as overly broad and failing to identify with specificity the type of information sought. Subject to

that objection, Centocor produced Deirdre Sanders, another attorney involved later in the prosecution, to testify on the topic. As Ms. Sanders did not author the two specific arguments that Abbott has picked out from the volumes of prosecution histories, she could not testify as to the bases for the arguments. And there is no evidence that Ms. Sanders had any role in drafting the arguments.

83. While Abbott now complains that Ms. Sanders was not prepared to testify about those two particular arguments, Abbott did not reasonably pursue the information it was looking for. First, Abbott chose not to seek a 30(b)(6) deposition on the topic of the two specific arguments made by Ms. Elmore in the 093, 861, and 102 applications. Second, Abbott chose not to seek additional 30(b)(6) testimony on those particular arguments following the deposition of Ms. Sanders. Third, Abbott chose not to depose the person that Abbott knew was the author of those arguments, Ms. Elmore. Given that Ms. Sanders's 30(b)(6) deposition took place well before the end of discovery, any of those options would have been available to Abbott if it really intended to determine the bases for Ms. Elmore's arguments and what information she had available to her at the relevant time.

84. Having failed to do any of those things, Abbott cannot now rely for its sole evidence of intent on the fact that Ms. Sanders did not know the bases for the arguments made years ago by Ms. Elmore. The absence of evidence on that point provides no basis to infer an attempt by Centocor to withhold information – it is instead the result of Abbott's failure to reasonably pursue that information in discovery. Put simply, Abbott has a burden to prove intent by clear and convincing evidence and Abbott's failure to seek discovery of relevant evidence on intent will not be used to draw an adverse inference under these circumstances.

85. Abbott has also failed to provide any evidence that the inventors were aware of the arguments made by Ms. Elmore. Abbott's assertion that the inventors were "heavily involved" in the patent prosecution is unsupported.

c) There Is Evidence Of Good Faith

86. Further countering Abbott's request that the Court infer deceptive intent, the evidence actually shows that the inventors and attorneys acted in good faith by ensuring that the Patent Office had the Moller paper, which provided the neutralization activity, affinity, and species specificity profile of MAK-195.

87. The inventors specifically point out the Moller paper in two different places in the specification of all the applications at issue. First, in the Background Section of the patent specification, the Moller paper is described as disclosing "murine mAbs specific for recombinant human TNF which had neutralizing activity in vitro" (PX 1 at 2:61-3:1; Ex. 11 at ABT01361964-65; Ex. 12 at ABT01362111; Ex. 13 at ABT01362809; Ex. 5 at ABT01364787-88; Ex. 14 at ABT01366684; Ex. 6 at ABT01364026; Ex. 4 at ABT01363015-16; Ex. 15 at ABT01365731; Ex. 16 at ABT01367227; Ex. 17 at ABT01367892; Ex. 18 at ABT01362274; Ex. 2 at CCOR00000174).

88. The inventors also cited the Moller paper in the section of each specification describing the species specificity profiles, stating:

> The ability of A2 or cA2 to react with TNF from different animal species was also evaluated. As mentioned earlier, there are multiple epitopes on human TNF to which inhibiting and/or neutralizing mAbs will bind (Moller, et al., infra). …
>
> [T]he epitope recognized by A2 is one shared by human and chimpanzee TNFα.

(PX 1 at 49:42-46, 49:60-61; Ex. 11 at ABT01362027-28; Ex. 12 at ABT01362167; Ex. 13 at ABT01362865; Ex. 5 at ABT01364871; Ex. 14 at ABT01366775-76; Ex. 6 at ABT01364109;

Ex. 4 at ABT01363099; Ex. 15 at ABT01365821; Ex. 16 at ABT01367318-19; Ex. 17 at ABT01367970; Ex. 18 at ABT01362339; Ex. 2 at CCOR00000251-52).

89. In addition to the two references to Moller in the specifications of each of the applications in the patent family, the Moller paper was also cited by applicants during prosecution of the priority applications in Information Disclosure Statements (Ex. 6 at ABT01364606; Ex. 4 at ABT01363673; Ex. 15 at ABT01366105; Ex. 5 at ABT01365160; Ex. 16 at ABT01367837; Ex. 14 at ABT01367101; Ex. 18 at ABT01362432; Ex. 2 at CCOR00000439).

90. Attorney Elmore herself pointed out to the patent examiner on multiple occasions that the Moller paper describes a "species specific murine monoclonal antibody, mAb 195, which neutralizes human TNF" (Ex. 4 at ABT01363698; Ex. 5 at ABT01365265-66; Ex. 6 at ABT01364628).

91. Further, the same Centocor report that Dr. Marks relied upon for binding information on cA2, also states the understanding in the Centocor technical group in 1997 that "none of the antibodies thus far studied individually bind to more than a fraction of the [cA2] epitope, making cA2 unique in its binding to TNFα" (DX 412 at CCOR00021300). It is now undisputed that this statement, made in an internal Centocor report, is consistent with the arguments made by Attorney Elmore to the PTO (Marks, Bench Trial at 117:12 – 118:3) (Ex. 7).

92. The Court therefore finds that Abbott has failed to prove by clear and convincing evidence that there was an intent to deceive the Patent Office during prosecution of the 1994 applications.

4. Abbott Has Failed To Prove A Necessary And Immediate Relation To The 775 And 239 Patents

93. As set forth above, the Court has found that Abbott failed to meet its burden of proving by clear and convincing evidence that inequitable conduct was committed during prosecution of any of the 1994 applications. But even if Abbott could have done so, the Court finds that Abbott has failed to meet its burden of proving that the 775 and/or 239 Patents in suit are somehow infected by that conduct. To this end, Abbott has failed to provide any evidence that the arguments made during the prosecution of the 093, 861, and 102 applications have an "immediate and necessary relation" to the enforceability of the 775 or 239 Patents as required by law.

94. Hundreds of different claims were pending at the time Attorney Elmore's arguments were made in the 093, 861, and 102 prosecutions, including claims to methods of treating Crohn's disease, claims to antibodies that bind to particular amino acid sequences, claims to neutralizing antibodies, claims to antibodies with particular affinities for TNF, and claims for antibodies that compete with A2 for binding to TNF (Ex. 4 at ABT01363162, 1363171, 1363606; Ex. 5 at ABT01365111; Ex. 6 at ABT01364572-73). Contrary to Abbott's assertion (D.I. 300 at 9), the competition with A2 element was not common to the rejected claims of those applications – most of the pending claims did not recite that limitation.

95. Abbott has offered no evidence that there is an immediate and necessary relation between: (a) the arguments made by Attorney Elmore directed to the claims pending in the 1994 applications; and (b) the claims in the 775 and 239 Patents, which have some, but not all (or even most) of the claim elements that were involved when the arguments were made. In the absence of evidence, particularly on issues regarding materiality, the Court declines to find that immediate and necessary relation.

5. Centocor Did Not Have A Sample Of MAK-195

96. Abbott contends that Centocor's failure to test MAK-195 is grounds for finding inequitable conduct. Because Abbott has failed to prove by clear and convincing evidence that Centocor had a sample of MAK-195, or any obligation to test it, the Court disagrees.

97. Dr. Salfeld, testifying as an Abbott corporate representative on the issue, was not aware of any sample of MAK-195 ever being provided to either Centocor or NYU (Salfeld, Bench Trial at 197:22 – 198:12) (Ex. 7).

98. So Abbott relies on a February 21, 1991 letter from Dr. Gert Riethmuller, a scientist at the Institute for Immunology at the University of Munich, to the President of Centocor as purportedly evidencing that Centocor had a MAK-195 sample (DX 80). As an initial matter, that letter was not authored by Centocor, and no witness has testified about its authenticity or the truth of the assertions therein. The letter is therefore entitled to little weight.

99. But, even taking it at face value, the letter does not mention the MAK-195 antibody, and the evidence suggests that Dr. Riethmuller was referring in his letter to a different antibody. Dr. Riethmuller wrote that he was sending an anti-TNF antibody of Knoll/BASF "which has successfully been used in GvH-disease" (DX 80). The Moller publication from 1990 reports that polyclonal anti-TNF antibodies had been used to treat GvH disease, but does not mention that the monoclonal antibody MAK-195, or for that matter any other monoclonal antibody, had been used to treat GvH disease (PX 192 at CCOR00011489). Thus, to the extent that the Riethmuller letter can be construed as indicating that he intended to send any antibody sample to Centocor, it only suggests that he intended to send a polyclonal antibody, not the monoclonal antibody MAK-195, to Centocor.

100. Abbott also relies on an undated "Data Sheet" that refers to an antibody identified as "monoclonal anti-TNFα antibody 195" (DX 79) as purportedly evidencing that Centocor, at

some undefined point in time, had a sample of MAK-195 in its possession. The document was not authored by Centocor, and no witness has testified about its authenticity or the truth of the assertions therein. Like the Riethmuller letter, the data sheet is therefore entitled to little weight.

101. The data sheet also provides little support for Abbott's claims that Centocor had a sample of MAK-195. Dr. David Shealy, testifying as Centocor's corporate representative on the issue, stated that he found no reference to MAK-195 in any other documents that he reviewed, that there was no date or signature on the data sheet that would indicate when it was received, that no one at Centocor who worked on the initial in-house testing of A2 and cA2 remembered receiving or testing the antibody, and that there was no record of any testing of the antibody in the lab notebooks of the employees who would have done such testing, if it was ever done (Shealy Dep. at 119:11 – 121:16) (Ex. 19).

102. Taken together, the evidence, including the testimony of Abbott's corporate representative, fails to show that Centocor ever had a sample of MAK-195.

103. Finally, whether or not a sample was received by Centocor, Abbott admits that Centocor never tested MAK-195 (D.I. 280 at 20-21). And, of course, Centocor had no obligation to do so. The Court therefore declines to adopt Abbott's contention that Centocor's failure to test MAK-195 is a basis for finding inequitable conduct.

C. Prosecution Laches

1. U.S. Application No. 07/670,827, filed on March 18, 1991 (abandoned)

104. The 775 Patent-in-suit originally claimed priority to twelve prior applications ("the 775 Patent family"), the earliest of which was filed on March 18, 1991 (PX 1). The 239 Patent-in-suit claims priority to the 775 Patent as a divisional application (PX 2).

105. In the first application in the priority chain, application serial number 07/670,827 (the "827 application") (Ex. 11), the PTO issued a Notice to File Missing Parts for this application on March 28, 1991 because the application was not submitted with the necessary filing fees and executed declarations of the inventors (*id*. at ABT01362045). Applicants paid the requisite filing fees and submitted the executed declarations on May 17, 1991 (*id*. at ABT01362046-49).

106. On April 8, 1992, the examiner imposed a restriction requirement on the 827 application, requiring election of a single invention for examination (*id*. at ABT01362050-53).

107. The April 8, 1992 restriction requirement was not responded to, and the examiner issued a Notice of Abandonment on November 17, 1992 (*id*. at ABT01362055).

2. U.S. Application No. 07/853,606, filed on March 18, 1992 (abandoned)

108. Meanwhile, before receiving the restriction requirement in the 827 application, applicants filed a second application (application serial number 07/853,606, the "606 application") as a continuation-in-part of the 827 application on March 18, 1992 (Ex. 12 at ABT01362086-87). A continuation-in-part application is an application filed during the pendency of the parent application and which adds new information to the specification (Steiner Bench Tr. at 143:22-144:4) (Ex. 7). The PTO issued a Notice to File Missing Parts for this application on April 22, 1992 because the application was not submitted with the necessary filing fees and executed declarations of the inventors (Ex. 12 at ABT01362206). Applicants paid the requisite filing fees and submitted the executed declarations on May 22, 1992 (*id*. at ABT01362208-215).

109. The specification of the 606 application was amended to add new information, including an example describing clinical activity and efficacy of the cA2 antibody of the invention (*id*. at ABT01362184-186).

110. On March 19, 1993, the Patent Office issued a Communication to the applicant, informing the applicant that the application did not comply with certain formalities relating to sequence listings (*id*. at ABT01362217). Applicants did not respond to this Communication, and on September 22, 1993, the examiner issued a Notice of Abandonment (*id*. at ABT01362219).

3. U.S. Application No. 07/943,852, filed on September 11, 1992 (abandoned)

111. Six months after the 606 application was filed, applicants filed their third application – application serial number 07/943,852 (the "852 application") (Ex. 18 at ABT01362242). The 852 application was filed on September 11, 1992 as a continuation-in-part of the 606 application (*id*. at ABT01362243). The PTO issued a Notice to File Missing Parts for this application on September 28, 1992 because the application was not submitted with the necessary filing fees and executed declarations of the inventors (*id*. at ABT01362395). Applicants paid the requisite filing fees and submitted the executed declarations on November 10, 1992 (*id*. at ABT01362397-399).

112. The specification of the 852 application was amended to add new information to the specification, including an example describing the results of a clinical study of the use of an antibody of the invention to treat rheumatoid arthritis (*id*. at ABT01362356-367).

113. On May 6, 1993, applicants telephonically elected to prosecute one group of claims in the 852 application (*id*. at ABT01362407). On June 23, 1993, an Office Action was issued in which all the claims were rejected for a variety of reasons, including lack of enablement (*id*. at ABT01362404-430).

114. Applicants did not respond to this Office Action, and on January 24, 1994, the examiner issued a Notice of Abandonment (*id*. at ABT01362435).

4. U.S. Application No. 08/013,413, filed February 2, 1993 (abandoned)

115. Less than five months after the 852 application was filed, applicants filed another continuation-in-part application. On February 2, 1993, application serial number 08/013,413 (the "413 application") was filed as a continuation-in-part of the 852 application (Ex. 13 at ABT01362778).

116. The specification of the 413 application was amended to add new information to the specification, including an example describing the results of a clinical study of the use of an antibody of the invention to treat Crohn's Disease (*id*. at ABT01362892-894).

117. The PTO issued a Notice to File Missing Parts for this application on March 19, 1993 because the application was not submitted with the necessary filing fees and executed declarations of the inventors (*id*. at ABT01362919). Applicants paid the requisite filing fees and submitted the executed declarations on May 12, 1993 (*id*. at ABT01362923-927).

118. On August 12, 1993, the Patent Office issued a Communication to the applicant, informing the applicant that the 413 application did not comply with certain formalities relating to sequence listings (*id*. at ABT01362928). A corrected sequence listing was submitted less than one month later, on September 8, 1993 (*id*. at ABT01362931-942).

119. On September 27, 1993, applicants telephonically elected to prosecute one group of claims in the 413 application (*id*. at ABT01362948). On October 27, 1993, an Office Action was issued in which all the claims were rejected for a variety of reasons, including lack of enablement (*id*. at ABT01362943-973).

120. Applicants did not respond to this Office Action and a Notice of Abandonment was issued on May 25, 1994 (*id*. at ABT01362979).

5. U.S. Application No. 08/010,406, filed January 29, 1993 (abandoned)

121. Application serial number 08/010,406 (the "406 application") was filed on January 29, 1993, but did not claim priority to any of the other prior-filed applications (Ex. 20 at ABT01362460).

122. The PTO issued a Notice to File Missing Parts for this application on March 9, 1993 because the application was not submitted with the necessary filing fees and executed declarations of the inventors (*id*. at ABT01362546). Applicants paid the requisite filing fees and submitted the executed declarations on April 8, 1993 (*id*. at ABT01362548-552).

123. On June 30, 1993, the Patent Office issued a Communication to the applicant, informing the applicant that the 406 application did not comply with certain formalities relating to sequence listings (*id*. at ABT01362553). A corrected sequence listing was submitted on July 29, 1993 (*id*. at ABT01362554-583).

124. A restriction requirement was imposed on the 406 application on January 11, 1994, requiring restriction to one of two groups of inventions (*id*. at ABT01362593-597). Applicants elected claims of Group I less than one month later, on January 31, 1994 (*id*. at ABT01362634-635).

125. An Office Action was issued on March 28, 1994, in which all the pending claims were rejected for a variety of reasons (*id*. at ABT01362636-643). Applicants submitted amendments and arguments in response to the Office Action on September 28, 1994 (*id*. at ABT01362663-674).

126. A second Office Action was issued on January 25, 1995 in which all the pending claims were again rejected (*id*. at ABT01362680-690). This application went abandoned for failure to respond to the Office Action.

127. The Court ruled on May 27, 2009 that Centocor was estopped from asserting that the pre-February 1994 applications complied with the first paragraph of 35 U.S.C. § 112 in order to gain benefit of the filing dates of those applications (D.I. 233 at 15, as amended, D.I. 234). The Court therefore does not consider the prosecution of applications prior to those filed February 4, 1994, or any of the delays alleged by Abbott, to be relevant to considering prosecution laches. If, however, this prosecution is considered, the Court does not find any of the actions to be unreasonable or to introduce undue delay.

6. U.S. Application No. 08/192,093 (U.S. Pat. No. 6,284,471), U.S. Application No. 08/192,102 (U.S. Pat. No. 5,656,272) and U.S. Application No. 08/192,861 (U.S. Pat. No. 5,919,452), filed on February 4, 1994 (collectively "the 1994 applications")

128. Applicants filed three separate applications on February 4, 1994: application serial numbers 08/192,093 (the "093 application"), 08/192,102 (the "102 application"), and 08/192,861 (the "861 application") (Ex. 4 at ABT01363000-181; Ex. 6 at ABT01363978-4187; Ex. 5 at ABT01364745-952). Each application was a continuation-in-part of the 413 application (Ex. 4 at ABT01363013; Ex. 6 at ABT01363979; Ex. 5 at ABT01364746). Information was added to the specification of each application, including, inter alia, examples describing the results of clinical studies of the use of an antibody of the invention to treat sepsis, arthritis, and severe ulcerative colitis (Ex. 4 at ABT01363117, ABT01363131-160; Ex. 6 at ABT01364127, ABT01364141-170; Ex. 5 at ABT01364889, ABT01364903-932). Disclosure of human antibodies as well as the citation of numerous references for making human antibodies was added to each specification (Ex. 4 at ABT01363044, lines 25-28, ABT01363021, lines 10-12,

ABT01363044, lines 12-20; Ex. 6 at ABT01364054, lines 27-30, ABT01364054, lines 13-22, ABT01364031, lines 11-13; Ex. 5 at ABT01364816, lines 25-28, ABT01364816, lines 12-20, ABT01364793, lines 10-12).

129. The 093 application issued as U.S. Patent No. 6,284,471 on September 4, 2001 (DX 475). The claims of the 471 patent are generally related to chimeric anti-TNFα antibodies having particular sequences (*id*. at col. 97-98).

130. The 102 application issued as U.S. Patent No. 5,656,272 on August 12, 1997 (DX 471). The claims of the 272 patent are generally directed to methods of treating Crohn's disease using chimeric anti-TNFα antibodies (*id*. at col. 97-98).

131. The 861 application issued as U.S. Patent No. 5,919,452 on July 6, 1999 (DX 473). The claims of the 452 patent are generally directed to methods of treating TNFα-mediated diseases using chimeric anti-TNFα antibodies (*id*. at col. 99-100).

7. U.S. Application No. 08/324,799 filed October 18, 1994 (U.S. Pat. No. 5,698,195)

132. On October 18, 1994, while each of applications 093, 102, and 861 were pending, application serial number 08/324,799 (the "799 application") was filed as a continuation-in-part of the 093, 861, and 102 applications (Ex. 15 at ABT01365670-914). The 779 application issued as U.S. Patent No. 5,698,195 on December 16, 1997, just a few months after the 272 patent issued, and more than three years before the 471 patent would issue (DX 472). The claims of the 195 patent are generally directed to methods of treating rheumatoid arthritis using chimeric anti-TNFα antibodies (*id*. at col. 107-110).

8. U.S. Application No. 08/570,674, filed December 11, 1995 (abandoned)

133. On December 11, 1995, again while each of applications 093, 102, and 861 were pending, and before any of the 1994 applications issued as patents, application serial number

08/570,674 (the "674 application") was filed as a continuation-in-part of the 799 application (Ex. 14 at ABT01366639-863). The claims pending in the 674 application would eventually be canceled and added to the 093 application (see Ex. 14 at ABT01367197 and Ex. 4 at ABT01363767). In doing so, the 674 application was then abandoned (Ex. 14 at ABT01367200).

9. U.S. Application No. 09/133,119, filed on August 12, 1998 (U.S. Pat. No. 6,277,969

134. While still prosecuting the 093 application, another application was filed in the family. Application serial number 09/133,119 (the "119 application") was filed on August 12, 1998 as a division of the 674 application (Ex. 16 at ABT01367210-405). The 119 application issued as U.S. Patent No. 6,277,969 on August 21, 2001, a few weeks before the 471 patent would issue (DX 474).

10. U.S. Application No. 09/756,398, filed on January 8, 2001 (U.S. Pat. No. 6,835,823)

135. While both the 119 and 093 applications were pending, application serial number 09/756,398 (the "398 application") was filed on January 8, 2001 as a continuation of the 119 application (Ex. 17 at ABT01367868-8055). The 398 application issued as U.S. Patent No. 6,835,823 on December 28, 2004 (DX 477).

11. U.S. Application No. 10/198,845, filed on July 18, 2002 (U.S. Pat. No. 7,070,775

136. During the pendency of the 398 application, application serial number 10/198,845 (the "845 application") was filed as a continuation of the 398 application on July 18, 2002 (Ex. 2 at CCOR00000097-325). The 845 application issued on July 4, 2006 as U.S. Patent No. 7,070,775 (PX 1).

12. U.S. Application No. 11/314,941, filed on December 20, 2005 (U.S. Pat. No. 7,276,239)

137. During the pendency of the 845 application, application serial number 11/314,941 (the "941 application") was filed as a division of the 845 application on December 20, 2005 (Ex. 3 at CCOR00019962-20120). The 941 application issued on October 2, 2007 as U.S. Patent No. 7,276,239 (PX 2).

13. Prosecution of the 775 Patent Family

a) Centocor Did Not Unreasonably Delay in Prosecuting the 775 Patent Family

138. Prosecution of the 775 and 239 Patents and the patents and applications within the 775 Patent family was reasonable and consistent with practices in prosecution of life science patents.

139. Abbott presented no evidence regarding what practices are considered reasonable in the prosecution of life sciences patents. Its expert, Arthur Steiner, testified that he has very limited experience in prosecuting applications in the biotechnology field (Steiner, Bench Trial at 156:8-23) (Ex. 7). He also testified that as an examiner with the USPTO, he did not examine patent applications in the biotechnology field (*id*. at 156:24 - 157:4). Mr. Steiner admitted that he had no experience in evaluating: (1) the extent to which applicants sought extensions of time in the biotechnology field (*id*. at 157:16-21, 158:14-20); (2) the extent to which applicants filed continuation-in-part applications and abandoned them (*id*. at 157:22 - 158:3, 158:10-13); and (3) the extent to which claims may surface years after a priority application was filed (*id*. at 158:4-9). He also admitted that he had no understanding of whether the length of time between filing an application and issuance or abandonment of the application was any longer in the biotechnology field as compared to other technology fields (*id*. at 158:21 - 159:1). Mr. Steiner

was thus forced to admit that he could not say whether the prosecution of the 775 Patent was typical for patents in the biotechnology field (*id*. at 159:8-11).

140. In contrast, Centocor's expert, Gerald Murphy, has prosecuted life sciences patents for 30 years (Murphy, Bench Trial at 208:22 - 209:5) (Ex. 7). Mr. Murphy has prosecuted approximately 1500 applications in the biotechnology field (*id*. at 209:10-15). He testified that, based on his experience, the 775 patent family was prosecuted with reasonable diligence (*id*. at 209:16-23), and that the prosecution was consistent with the general practice in the life sciences area (*id*. at 209:24 - 210:7).

141. In prosecuting patents in the biotechnology field, the general goal is to get the broadest protection possible for the disclosed invention, which includes seeking different types of broad claims (such as method claims and product claims) (*id*. at 210:11 - 211:8). It is also important to seek narrow claims that cover the invention that encompasses a developed pharmaceutical product (*id*. at 211:9-21; Townsend Dep. at 305:25 - 306:23 (Ex. 21)). That general approach was followed in the 775 Patent family, namely that claims to Centocor's commercial cA2 antibody and methods of its use were pursued first (Townsend Dep. at 306:25 - 307:20) (Ex. 21).

142. It is common in the field of biotechnology to have a series of continuations, divisionals, and continuation-in-part applications in the same patent family (Murphy, Bench Trial at 213:1-7) (Ex. 7). In particular, continuation-in-part applications are often filed to incorporate new clinical data that may be generated after the initial filing to support the patentability of the invention. Centocor filed continuation-in-part applications to add information learned about the invention as its research continued. For example, the 1994 applications included additional information about human antibodies and how they are made, as well as the

results of clinical trials reflecting the use of an antibody of the invention to treat various diseases (Townsend Dep. at 303:2 - 304:12 (Ex. 21); Ex. 4 at ABT01363044, lines 25-28; ABT01363021, lines 10-12, ABT01363044, lines 12-20, ABT01363117, ABT01363131-160; Ex. 6 at ABT01364054, lines 27-30, ABT01364054, lines 13-22, ABT01364031, lines 11-13, ABT01364127, ABT01364141-170; Ex. 5 at ABT01364816, lines 25-28, ABT01364816, lines 12-20, ABT01364793, lines 10-12; ABT01364889, ABT01364903-932).

143. In order to claim priority to an earlier application (to gain the benefit of the earlier application's filing date), a subsequent application must be filed while the earlier application is still pending, *i.e.*, there must be co-pendency between the applications (Murphy, Bench Trial at 214:13 - 215:4) (Ex. 7). A subsequent application can be filed as late as on the day an earlier application either issues as a patent or goes abandoned, and still have co-pendency. In 775 Patent family, Centocor filed each subsequent application in the family well in advance of a previous application either issuing or going abandoned. For example, the 606 application was filed one year after the 827 application was filed, and before the first office action was received in the 827 application. The 606 application was filed nearly eight months before the 827 application abandoned. Centocor chose to file three applications on the same day in February 1994, instead of prosecuting each of them serially. The applications were filed at least two months before the prior application (the 413 application) went abandoned. During the prosecution of the 1994 applications, Centocor filed and prosecuted four other applications, which resulted in the issuance of three patents.

144. Abbott argues that Centocor failed to speed abandonment of five applications, and that such failure contributes to a find of prosecution laches. Those five abandoned applications were filed prior to February 4, 1994. The Court has previously ruled that Centocor can not claim

priority before 1994. Even if the prosecution of the abandoned applications somehow contributed to a delay, which they did not, the prosecution of any of the applications filed prior to February 4, 1994 is not relevant to the 775 Patent's prosecution.

145. Abbott also argues that Centocor failed to file complete applications, but the filing of an incomplete application does not necessarily delay prosecution (Steiner, Bench Trial at 168:1-5) (Ex. 7). To assess whether the filing of an incomplete application actually delays prosecution depends on how many cases were pending before an examiner so as to determine whether there was a backlog of cases. (*id*. at 168:6-15). Abbott presented no evidence as to whether any of the incomplete Centocor applications actually resulted in a delay of prosecution (*id*. at 168:16-20).

146. Abbott also complains that Centocor's requests for extensions of time during the prosecution of the 775 Patent family led to a delay in prosecution. The maximum period of time for replying to an office action is set by statute and is usually six months (*id*. at 153:11-13). But the patent office sets a shortened statutory period of time of three months to reply to an office action (*id*. at 153:14-15). An applicant can petition for an extension of the shortened statutory period by paying a fee (*id*. at 153:15-16). It is common to file an extension of time in the pharmaceutical or biotech field, and especially when dealing with complex technical and legal issues (Murphy, Bench Trial at 226:24 - 227:8 (Ex. 7); Townsend Dep. at 286:3 - 288:24 (Ex. 21)). Extensions of time do not necessarily delay the prosecution of an application. Taking extra time to draft a more persuasive and complete response to an office action can result in getting an earlier notice of allowance and actually shorten prosecution (Murphy, Bench Trial at 228:6-19 (Ex. 7); Sanders Dep. at 96:19 - 97:5 (Ex. 22); Townsend Dep. at 286:3 - 288:24 (Ex. 21)).

147. Further, most of the extensions of time requested by Centocor that Abbott alleges introduced delay, could not delay prosecution of the 775 or 239 Patents because the extensions were taken after a subsequent continuation-in-part application was already filed. For example, during prosecution of the 093 application, nine requests for an extension of time were taken, but all of the requests for extension of time were taken after the next application in the chain – the 799 application – was already filed. Abbott's expert Mr. Steiner was thus forced to admit that the prosecution of the 093 application did not delay the prosecution of the 799 application (Steiner, Bench Trial at 169:10-23) (Ex. 7).

148. Abbott argues that prosecution was also delayed due to the filing of incomplete drawings or sequence listing in three applications. Two of the incomplete submissions occurred in applications predating the filing of the 1994 applications (Ex.12 at ABT01362217; Ex. 13 at ABT01362928). Prosecution predating the filing of the 1994 applications is irrelevant to the determination of prosecution laches. But, even if it were, Abbott has presented no evidence that the filing of incomplete applications results in the delay of prosecution.

149. Abbott's final argument is that Centocor unreasonably delayed the presentation of claims directed to human antibodies until it learned of Abbott's human antibody product, Humira. But Centocor did not use delay tactics in presenting claims directed to human antibodies, and instead prosecuted the applications in the 775 Patent family consistent with practice in the life sciences area, whereby Centocor first sought protection of its commercial product and methods of using the product, and later sought protection on other aspects of its invention (Townsend Dep. at 306:25 - 307:20) (Ex. 21). In July 2002, Centocor filed the 845 application with claims directed to chimeric and human anti-TNFα antibodies. By this time, Centocor had begun development of its own human anti-TNF antibody, CNTO-148, which was

later commercialized as Simponi™ (Scodari, Day 1 AM at 110:2-13) (Ex. 23). It wasn't until August 2005 that Centocor conducted competition testing with Humira and cA2 to determine whether the pending claims directed to human antibodies with cover Humira (Shealy Dep. at 282:19 - 283:13 (Ex. 19); DX 92). After finding that Humira and cA2 compete for binding as required by the pending claims, Centocor notified the patent office that the claims cover Humira (Ex. 2 at CCOR00000417). And, as Abbott patent attorney John Conway testified, it is not unusual or improper to draft claims to cover a competitor's product, as long as there is a basis in the pending application (Conway, Bench Trial at 205:15-25) (Ex. 7).

150. Abbott points to three Abbott press releases as evidence of Centocor's knowledge that Abbott had developed a fully human antibody by the spring of 2002 (Steiner, Bench Trial at 150:25 - 151:10 (Ex. 7); DX 53; DX 762; DX 763). There is no evidence that anyone at Centocor ever saw these press releases at any time, or knew that Abbott has developed its own human anti-TNFα antibody. There is no evidence that Abbott's development of a human anti-TNFα antibody in any way prompted Centocor to file claims covering human anti-TNFα antibodies in July 2002.

151. Accordingly, the Court finds that Abbott has not met its burden of proving by clear and convincing evidence that there was any unreasonable delay in prosecuting the patents in the 775 Patent family.

b) Abbott Was Not Prejudiced by Any Alleged Delay

152. Abbott asserts that it has been prejudiced by the alleged prosecution delay by Centocor of its human antibody claims because BASF AG refuses to indemnify Abbott for any litigation-related expenses. Abbott purchased BASF Pharma, the U.S. pharmaceutical group of BASF AG, in December 2000 (Poulos, Bench Trial at 174:22 - 175:1) (Ex. 7). BASF Pharma

developed the human antibody D2E7, which is the antibody in Abbott's Humira product (*id*. at 174:16-18).

153. As part of the acquisition of BASF Pharma, BASF AG agreed to reimburse Abbott for a portion of royalties Abbott pays to third parties for the sale of Humira under certain conditions. The agreement specifies that BASF AG must reimburse Abbott "for 50 percent of any and all expenses, including attorneys fees incurred in connection with the defense of any claim, action, complaint, cause of action, or proceeding commenced or threatened based upon, arising out of, or related to the allegation that the manufacture, use or sale of D2E7 by [Abbott] infringes patents or patent applications published on the date of [the] agreement" (*id*. at 179:10-20, referring to DX 836[2]).

154. Abbott has requested that BASF honor the indemnification provision and reimburse Abbott for its expenses associated with the present litigation (*id*. at 180:6-23; DX 806). BASF has refused to indemnify Abbott on the basis that the present litigation falls outside of the indemnity agreement (Poulos, Bench Trial at 180:24 - 181:21; DX 803). Abbott disagrees with this conclusion, and has urged BASF to honor the indemnity agreement reasoning that since the 775 Patent claims priority to a series of applications dating back to March 1991, it is covered by the agreement (Poulos, Bench Trial at 182:14 - 183:12, 192:2-11 (Ex. 7); DX 804).

155. But Abbott has yet to fully pursue the issue of indemnification with BASF (Poulos, Bench Trial at 192:12-21) (Ex. 7), and Abbott has not, but could, bring a lawsuit against BASF to require them to honor the indemnification provision (*id*. at 193:8-13). Accordingly, the Court finds that, even if the lack of BASF indemnification could be a basis for Abbott to claim

[2] Centocor has not attached DX 836 as an exhibit hereto because the Court has ordered that document sealed. Centocor will provide a copy of DX 836 to the Court if so requested.

prejudice, at this point, that claim is only speculative. Abbott has not proven that it has been or will be prejudiced by any actions of Centocor.

156. Abbott further contends that it conducted due diligence at the time it acquired BASF Pharma and did not unearth the Centocor patents. It argues that if it has discovered the Centocor patents, that discovery would have caused it to change the financial considerations of what it would have paid for BASF Pharma (*id*. at 189:22 - 190:13). But Abbott representative John Poulos testified at trial that while he was responsible for running the due diligence team before acquiring BASF, he did not consider the Centocor patents or products (*id*. at 193:20 - 194:13).

157. Abbott also alleges that it has spent "well over 100 million dollars" on clinical trials and pre-launch marketing expenses for Humira from the time it acquired BASF Pharma in 2000 until the publication of the 775 Patent in July 2003 (*id*. at 191:8-16) and suggests that it is prejudiced because it expended this money without appreciating the full scope of Centocor's invention.

158. But once the 272 patent in the family issued and published, all of the previous applications within the prosecution chain became available to the public (Steiner, Bench Trial at 144:15 - 145:25) (Ex. 7). So, by no later than August 12, 1997, when the 272 patent issued, the public was put on notice that the specification of the 272 patent included disclosure of an invention directed to human anti-TNFα antibodies.

159. That means that, by 1997, anyone looking at the Centocor patent would have known that the summary of the invention section of the patent included human anti-TNF antibodies (Poulos, Bench Trial at 194:20-25) (Ex. 7). Patents with the same disclosure

continued to issue in December 1997 and July 1999, *i.e.*, before Abbott entered into the agreement with BASF AG to acquire BASF Pharma (DX 472; DX 473).

160. The Court finds that Abbott's failure to investigate or consider the full scope of Centocor's invention before acquiring BASF and investing money in clinical trials and other marketing expenses relating to Humira is not evidence of prejudice caused by Centocor.

161. Abbott also argues that it has been prejudiced because, had it known of Centocor's human antibody claims in 2002, it could have sought a license during negotiations with Centocor in 2002. But Abbott presented no evidence to support this type of prejudice, and in any event, this argument is inconsistent with Abbott's prior statements to the Court. In requesting a stay of proceedings to pursue an arbitrations against Centocor, Abbott told the Court in 2007 that it had already gotten a license from Centocor in the 2002 negotiations (D.I. 28 at 1). Abbott told the Court that it "has asserted that it has an express and/or implied license to the '775 patent …" (*id.*). If Abbott believed it was licensed in 2007, before an arbitrator ruled otherwise, it must have had that belief in 2002. Its suggestion that, had it known about the Centocor patents in 2002, it would have asked for a license, is not credible.

162. Abbott also argues that it sustained evidentiary prejudice because of Centocor's alleged delay. But here too, Abbott has presented no evidence to support that it suffered from any evidentiary prejudice. Abbott suggested that Centocor's alleged delay prevented it from obtaining a viable sample of the prior art antibody CDP-571 in an attempt to invalidate the 775 Patent. Not only did Abbott present no evidence that proves it was prejudiced by any delay by Centocor, Abbott itself bears the responsibility for failing to prove the authenticity of the CDP-571 antibody. Abbott noticed the deposition of UCB, the company who owns the antibody, but Abbott chose not to take that deposition (Ex. 24). Then Abbott waited until after the close of

discovery before producing a declaration from a UCB representative in an attempt to authenticate the sample and verify its viability. Judge Everingham properly refused to admit that declaration before trial, and the Court made the same ruling during trial (Day 4 AM at 123:6 - 125:2) (Ex. 23). The Court, accordingly, finds that Abbott has not proven that it sustained any evidentiary prejudice due to Centocor's alleged delay in prosecution.

163. Finally, any alleged delay in prosecuting the 775 Patent actually worked to Abbott's benefit and Centocor's detriment. The 775 Patent will expire 20 years from the filing of the earliest application in the family on March 18, 2011, *i.e.*, 20 years from March 18, 1991 (Murphy, Bench Trial at 233:20-25) (Ex. 7). Centocor could only collect damages for Abbott's infringement of the 775 Patent from the date it issued on July 4, 2006. Had the prosecution of the 775 Patent ended earlier, Centocor could have collected millions if not billions of additional dollars from the sale of Humira (*id*. at 233:20 - 234:14). As a matter of equity, the Court finds that this weighs substantially against Abbott on this issue.

164. Accordingly, the Court finds that Abbott has not met its burden of proving by clear and convincing evidence that it was prejudiced by any alleged delay in the prosecution of the 775 Patent family. Abbott has also failed to meet its burden of proving by clear and convincing evidence that the 775 or 239 Patents, or any patent in the 775 patent family, should be held unenforceable under the doctrine of prosecution laches.

III. INDEFINITENESS

165. Abbott contends that the "competitively inhibits" limitation of the claims of the 775 and 239 Patents is indefinite. But, Abbott, the party bearing the burden of proof on indefiniteness, presented ***no evidence*** at the trial on the issue, from its expert Dr. Marks or otherwise. Indeed, Abbott chose not to offer testimony from Dr. Marks on the issue, so that he would not be subjected to cross-examination based on his prior testimony. And Abbott chose not

to offer at trial any deposition testimony from other witnesses to support its indefiniteness defense.

166. The evidence adduced during the jury trial shows, contrary to Abbott's unsupported allegations, that those skilled in the art would understand the bounds of the asserted claims, particularly regarding the test conditions used to show competitive inhibition. Abbott's expert, Dr. Marks, actually relied on antibody competition testing, done by a different retained expert, to support his conclusions about invalidity in this case. Dr. Marks "played no role in the development of the protocol" used in those tests (Marks, Day 3 PM at 60:18-21) (Ex. 23) and never even spoke to the scientist who set up and performed those tests (*id*. at 57:24 – 58:2). Nonetheless, Dr. Marks had no difficulty relying on those tests, which were "the kind of test that [he] would normally rely on" (*id*. at 58:12-15).

167. Abbott's expert Dr. Marks also had ***no*** problems with the protocols used by Centocor scientist Susan Tam to show antibody competition or the quality of her results:

> Q: And, in fact – I mean, you don't have any problems with the protocols that [Ms. Tam] used to generate her data, right?
>
> A: ***Not at all.***
>
> Q: And you don't question the period of incubation that Ms. Tam used when she let the antibody sit in the well with TNF, right?
>
> A: ***I do not.***
>
> Q: And you do not question the data that Ms. Tam generated, right?
>
> A: ***I do not.***
>
> Q: In fact, you don't have any problem with the quality of the data generated by Ms. Tam, right?
>
> A: The quality is good.

(Marks, Day 3 AM at 149:3-16) (Ex. 23).

168. Dr. Marks did argue that the "direction" of the competition assay may matter, but he failed to present any credible evidence to support his argument. Indeed, the only evidence that Dr. Marks relied on in support of his hypothesis was a single publication – the Moller paper (*id*. at 150:8 – 151:4). But that paper does not support his position. On cross examination, Dr. Marks admitted that the Moller paper does ***not*** indicate that there is "one-way" competition (*id*. at 151:5-20). And Centocor's expert, Dr. Adams, testified that the "direction" of the test does not matter for these antibodies, meaning that the results would be the same in either direction (Adams, Day 1 PM at 107:17 – 108:2) (Ex. 23).

169. Furthermore, Dr. Marks identified ***no*** competition testing for Humira and A2 that gave a different result than Ms. Tam's or that suggested that Ms. Tam's results were somehow incorrect (Marks, Day 3 AM at 149:17-20) (Ex. 23). There is, therefore, no evidence that different assays with these antibodies yield different results.

170. The Court, accordingly, finds that Abbott has failed to prove by clear and convincing evidence that the claim term "competitively inhibits" is indefinite.

PROPOSED CONCLUSIONS OF LAW

I. JURISDICTION AND VENUE

1. This is an action for patent infringement arising under the patent laws of the United States, 35 U.S.C. § 101, et. seq. This Court has jurisdiction under 28 U.S.C. §§ 1331 and 1338(a), and venue lies in this district under 28 U.S.C. §§ 1391 (b)-(c) and 1400(b).

II. GOVERNING LAW

2. In patent cases such as this one, the law of the Federal Circuit governs the substantive law to be applied. 28 U.S.C. § 1295.

III. ABBOTT'S BURDEN OF PROOF

3. For Abbott to prevail on its defense that the 775 Patent in unenforceable due to inequitable conduct, Abbott must prove the facts underlying this defense by clear and convincing evidence. *Star Scientific, Inc. v. R.J. Reynolds Tobacco Co*., 537 F.3d 1357, 1365 (Fed. Cir. 2008).

4. For Abbott to prevail on its prosecution laches defense, it must show that the 775 Patent issued after an unreasonable and unexplained delay in prosecution. *Symbol Techs., Inc. v. Lemelson Med., Education & Research Foundation,* 277 F.3d 1361, 1364-68 (Fed. Cir. 2002); *accord In re Bogese*, 303 F.3d 1362, 1367 (Fed. Cir. 2002). The Federal Circuit has not articulated the burden of proof that a challenger must meet to prevail on a prosecution laches defense, and district courts who have decided the issue of prosecution laches have either not articulated a standard or have decided the issue under a preponderance of the evidence. *Intuitive Surgical, Inc. v. Computer Motion, Inc*., No. 01-203-SLR, 2002 U.S. Dist. LEXIS 24808, *16, n. 4 (D. Del. Dec. 10, 2002) (Ex. 31). Under either a clear and convincing burden or under a preponderance of the evidence, Abbott has failed to carry its burden of proving that this is an unusual and egregious case that justifies holding the patents unenforceable for prosecution laches.

5. For Abbott to prevail on its indefiniteness defense, it must prove "by clear and convincing evidence that a skilled artisan could not discern the boundaries of the claim based on the claim language, the specification, and the prosecution history, as well as her knowledge of the relevant art area." *Halliburton Energy Servs., Inc. v. M-I LLC*, 514 F.3d 1244, 1249-50 (Fed. Cir. 2008); *see also Intel Corp. v. VIA Techs., Inc.*, 319 F.3d 1357, 1366 (Fed. Cir. 2003) ("Any fact critical to a holding on indefiniteness … must be proven by the challenger by clear and convincing evidence.").

IV. INEQUITABLE CONDUCT

6. To prove its defense of inequitable conduct, Abbott must show that Centocor made an affirmative misrepresentation of material fact, failed to disclose material information, or submitted material false information to the Patent Office, and did so with an intent to deceive the Patent Office. *Star Scientific*, 537 F.3d at 1365.

7. Materiality and intent are separate and essential elements of an inequitable conduct defense, and both elements must be proven by clear and convincing evidence. *Id.*; *M. Eagles Tool Warehouse, Inc. v. Fisher Tooling Co*., 439 F.3d 1335, 1340 (Fed. Cir. 2006). If Abbott is able to carry its burden as to both materiality and intent, the Court must then carefully balance the equities to determine whether Centocor's alleged conduct before the PTO was so egregious as to compel a finding that the 775 and/or 239 Patents is unenforceable. *Star Scientific*, 537 F.3d at 1365.

8. Abbott must prove that Centocor acted with the "specific intent to deceive the PTO." *Id.* at 1366. In the absence of direct evidence, Abbott asks the Court to infer that intent, which requires that "the inference must not only be based on sufficient evidence and be reasonable in light of that evidence, but it must also be the ***single most reasonable*** inference able to be drawn from the evidence." *Id*. (emphasis added). Importantly, Centocor "need not offer any good faith explanation unless [Abbott] first [carries its] burden to prove a threshold level of intent to deceive by clear and convincing evidence." *Id*. at 1368. And, "[w]hen the absence of a good faith explanation is the only evidence of intent, … that evidence alone does not constitute clear and convincing evidence warranting an inference of intent." *M. Eagles*, 439 F.3d at 1341. Finally, the applicant's conduct must also be viewed in light of evidence of good faith. *Eaton Corp. v. Rockwell Int'l Corp.*, 323 F.3d 1332, 1345-46 (Fed. Cir. 2003).

9. The Court concludes that Abbott has failed to carry its burden of proving inequitable conduct.

10. First, Abbott has failed to provide clear and convincing evidence of any material misrepresentation or omission. As set forth above (Proposed Findings of Fact ¶¶ 37-67), the evidence at trial shows that both of Attorney Elmore's arguments were correct. First, it is true that it is unlikely that MAK-195 binds to the same epitope as the antibodies claimed. The evidence at trial actually shows that the epitopes are different. Second, it is true that one skilled in the art would not conclude that MAK-195 (a murine antibody) is identical to the antibodies claimed at the time (which were chimeric). Indeed, Abbott has not even tried to prove otherwise. Third, it is also true that MAK-195 does not contain the features that Attorney Elmore was referring to in her argument – MAK-195 is not a chimeric antibody and was not shown by the Moller paper to have superior clinical results. Abbott has, therefore, failed to prove by clear and convincing evidence that Attorney Elmore's arguments were material misrepresentations.

11. Abbott has also failed to prove that Attorney Elmore's arguments were material. The Moller paper was before the patent examiner because the inventors had disclosed it in IDS submissions and in the patent applications themselves (Proposed Findings of Fact ¶¶ 87-89). The examiner, as one skilled in the art with the Moller paper before her, was free to accept or reject Attorney Elmore's arguments. *Innogenetics, N.V. v. Abbott Labs.*, 512 F.3d 1363, 1379 (Fed. Cir. 2008); *CFMT, Inc. v. YieldUp Int'l Corp.*, 349 F.3d 1333, 1341-42 (Fed. Cir. 2003); *Akzo N.V. v. U.S. Int'l Trade Comm'n*, 808 F.2d 1471, 1482 (Fed. Cir. 1986). An attorney is free to present arguments about a reference that is before the examiner without fear of committing inequitable conduct. *Rothman v. Target Corp.*, 556 F.3d 1310, 1328-29 (Fed. Cir. 2009); *Young v. Lumenis, Inc.*, 492 F.3d 1336, 1348-49 (Fed. Cir. 2007). Here, Attorney Elmore has done

nothing more that argue about what those of ordinary skill would conclude from the disclosure found in the Moller paper, which was placed before the examiner by Centocor. Her arguments were therefore not material statements that can form the basis for inequitable conduct.

12. Second, Abbott has also failed to provide clear and convincing evidence that Centocor withheld any material information from the examiner. Abbott suggested at trial that Centocor withheld information about the MAK-195 antibody that was known to Dr. Vilcek and disclosed by him to Centocor inventors Scott Siegel and Peter Daddona. The evidence shows that, on the contrary, Dr. Vilcek's knowledge about MAK-195 was limited to the information that was disclosed in the Moller paper. And that information was presented to the Patent Office, both in the form of the Moller paper itself and in Attorney Elmore's argument that the Moller paper described a "species specific murine monoclonal antibody, mAb 195, which neutralizes human TNF."

13. As set forth above (Proposed Findings of Fact ¶¶ 70-72), Dr. Vilcek wrote on August 1, 1990 that the Moller paper set forth the neutralization activity and species specificity of a group of antibodies, one of which was MAK-195. Those characteristics about MAK-195 were in the Moller paper, and they were not withheld from the Patent Office. Then, on August 15, 1990, Dr. Vilcek described species specificity testing on the A2 antibody. Again, that A2 species specificity was not withheld from the Patent Office. Dr. Vilcek also wrote that, based on the species specificity, A2 resembled that MAK-195, which again was apparent to the patent examiner, who had the A2 results and the Moller paper and made rejections based on that resemblance.

14. When he was shown the Moller paper, Dr. Vilcek testified that his understanding of MAK-195 was that "it was another ***murine*** monoclonal antibody against human TNF that had

the ability to – to ***neutralize*** human TNF" (Vilcek, Bench Trial at 200:24 – 201:4) (Ex. 7) (emphasis added). That is the extent of the information about MAK-195 known to Centocor and NYU, and that information was disclosed to the Patent Office.

15. Third, Abbott has failed to provide clear and convincing evidence that Centocor had a sample of MAK-195 (Proposed Findings of Fact ¶¶ 96-103). Abbott has admitted that Centocor did no testing of MAK-195 (Abbott Opening Br. at 20-21).

16. Furthermore, Centocor had no duty to test a sample of MAK-195 in these circumstances. This is not a case where an applicant did not cite material prior art of which he was aware. *Medtronic Vascular, Inc. v. Boston Scientific Corp.*, No. 2:06-CV-78, 2008 U.S. Dist. LEXIS 67819, at *11 (E.D. Tex. Aug. 29, 2008) (Ex. 25). Nor is it a case where "questionable information" provided by a client warranted further investigation by an attorney. *Brasseler, U.S.A. I, L.P. v. Stryker Sales Corp.*, 267 F.3d 1370, 1382-83 (Fed. Cir. 2001). There is no requirement for an applicant to obtain prior art samples and test them. Thus, there is no inequitable conduct here based on Centocor's failure to test MAK-195.

17. Fourth, Abbott further argues that Centocor withheld the rejections in the 093, 861, and 102 applications during the prosecution of later children applications that issued as the 775 and 239 Patents. Contrary to Abbott's argument, the evidence shows that those rejections, which all occurred prior to the filing of the continuing applications that issued as the 775 and 239 Patents, were before the examiner.

18. According to the Manual of Patent Examining Procedure (MPEP), "[i]n all continuing applications, the parent applications should be reviewed by the examiner for pertinent prior art" (MPEP § 904) (Ex. 26). In examining a continuing application, be it a continuation, divisional, or continuation-in-part application, an examiner will consider information that was

considered by the Patent Office in the parent applications, and the applicant is under no duty to resubmit that information (MPEP § 609.02) (Ex. 27). Thus, the history of the 093, 861, and 102 applications was part of the information considered by the patent examiner during the prosecution of the 775 and 239 Patents.

19. Consistent with that general practice set forth in the MPEP, the examiner in this case, during prosecution of the 775 and 239 Patents, referred to the parent applications on multiple occasions (Ex. 2 at CCOR00000454, 00000511; Ex. 3 at CCOR21052).

20. Because the past file history of the parent applications is before the examiner of a child application, failure to resubmit information in a continuing application that was cited or submitted in the past in a parent application is not inequitable conduct. *ADT Corp. v. Lydall, Inc*., 159 F.3d 534, 547 (Fed. Cir. 1998) ("it cannot be inequitable conduct … not to resubmit … the information that was cited or submitted in the parent application"); *eBay Inc. v. IDT Corp*., No. 08-cv-4015, 2009 U.S. Dist. LEXIS 75475, *7-*8 (W.D. Ark. Aug. 24, 2009) (Ex. 28). The fact that the rejections in this case were made in the ***past*** history of the pending applications makes this case unlike those where an examiner is not informed of events that are happening in a co-pending prosecution. *See, e.g., Larson Mfg. Co. v. Aluminart Prods. Ltd.*, 559 F.3d 1317, 1338-39 (Fed. Cir. 2009) (examiner not informed about rejection in co-pending application); *McKesson Info. Solutions, Inc. v. Bridge Med., Inc.*, 487 F.3d 897, 920-21 (Fed. Cir. 2007) (finding that applicant should have made an examiner aware of a rejection over substantially similar claims in a co-pending application); *Dayco Prods., Inc. v. Total Containment, Inc.*, 329 F.3d 1358, 1361, 1365-66 (Fed. Cir. 2003) (examiner not informed about a co-pending application). Here, the failure to resubmit the past history of the priority applications, which is presumed to have been considered according to Patent Office procedure, is not a material

omission. Even if it were, the failure to inform an examiner about rejections made in the same family of priority applications does not show intent to deceive the Patent Office. *Leviton Mfg. Co. v. Universal Sec. Instruments, Inc.*, 304 F. Supp. 2d 726, 753 (D. Md. 2004); *Pharm. Res., Inc. v. Roxane Labs., Inc.*, No. 03-3357, 2008 U.S. Dist. LEXIS 29590, at *38, 42-43 (D. N.J. Apr. 10, 2008) (Ex. 32).

21. Fifth, for the Court to hold the 775 and 239 Patents unenforceable, Abbott must prove that there is an "immediate and necessary relation" between those patents and the arguments made in the 093, 861, and 102 applications. *Consol. Aluminum Corp. v. Foseco Int'l Ltd.*, 910 F.2d 804, 810 (Fed. Cir. 1990). As set forth above, Abbott has failed to show such a relation between the claims that were pending when those arguments were made and the claims asserted here. For that additional reason, Abbott has failed to prove that the asserted claims are unenforceable for inequitable conduct.

22. Finally, as set forth above (Proposed Findings of Fact ¶¶ 68-92), Abbott has failed to prove by clear and convincing evidence that the inventors, attorneys, or anyone else associated with the prosecutions of the 775 or 239 Patents, or the earlier patent applications in the family, intended to deceive the Patent Office. Specifically, the Court concludes not only that there is no direct evidence of intent, but that there is no basis in this case for inferring intent. Deceptive intent is not, in this case, the "single most reasonable inference," *Star Scientific*, 537 F.3d at 1366, because, as set forth above, it is reasonable to infer that the inventor's discussion of species specificity related to animal models for FDA testing rather than to the antibody epitopes or features. Taking into account the absence of material omissions or misstatements, the absence of any evidence that the inventors or attorneys knew that any arguments were false or

misleading, and the evidence of Centocor's good faith in pointing Moller out to the Patent Office, the Court concludes that there is no inference of deceptive intent.

V. PROSECUTION LACHES

23. Abbott contends that the 775 and 239 Patents are unenforceable under the doctrine of prosecution laches. For Abbott to prevail on this defense, it must show that the 775 and 239 Patents issued after an unreasonable and unexplained delay in prosecution. *Symbol Techs.,* 277 F.3d 1364-68; *accord In re Bogese*, 303 F.3d at 1367. The Federal Circuit has cautioned that prosecution laches is a tool which has been used sparingly and in only the most egregious of cases. *Symbol Techs., Inc. v. Lemelson Med., Education & Research Foundation,* 422 F.3d 1378, 1385 (Fed. Cir. 2005).

24. Laches requires proof not only that a plaintiff delayed for an unreasonable and inexcusable length of time, but also that the delay operated to the prejudice or injury of the defendant. *A.C. Aukerman Co. v. R.L. Chaides Construction Co.*, 960 F.2d 1020, 1032 (Fed. Cir. 1992) (*citing Costello v. United States*, 365 U.S. 265, 282 (1961)). Prosecution laches also requires a showing of prejudice to intervening adverse rights. The Federal Circuit recognized the prejudice aspect of the doctrine in relying on the Supreme Court decision in *Crown Cork & Seal Co. v. Ferdinand Gutmann Co.*, 304 U.S. 159 (1938), which "ratified prosecution laches as a defense to an infringement action involving new claims issuing from divisional and continuing applications that prejudice intervening public rights." *Symbol Tech.*, 277 F.3d at 1364; see also *In re Katz Interactive Call Processing Patent Litigation*, No. CV 2:07-ML-01816-B-RGK, at 102-103 (C.D. Cal. Aug. 13, 2009) (Ex. 29); *Verizon Cal Inc. v. Ronald A. Katz Tech. Licensing, L.P.*, No. 01-CV-9871 (RGK) (RCx), 2003 U.S. Dist. LEXIS 23553, at *62-63 (C.D. Cal. Dec. 2, 2003) ("it appears that proof of 'intervening adverse public rights' is a requisite element of a successful prosecution laches defense") (Ex. 30). Damages or monetary losses merely

attributable to a finding of liability for patent infringement do not amount to the "economic prejudice" required to support a laches defense. *A.C. Aukerman*, 960 F.2d at 1033. Thus, Abbott bears the burden of proving that any alleged delay in prosecution by Centocor operated to the prejudice or injury of Abbott.

25. Prosecution laches has its origin in Supreme Court cases dating back to the 1920s. *Symbol Tech*., 277 F.3d at 1364. At that time, it was possible for patent applicants to delay issuance of patents in order to maintain their patent application in secrecy and in order to obtain a longer patent term. U.S. patent applications were maintained in secret until the patents issued, and a patent applicant could theoretically keep an application pending for many years and "surprise" an infringer or even an entire industry with a "submarine patent" which typically would have a patent term of seventeen years from issuance.

26. To address this potential for abuse, Congress twice amended the patent statute. First, in 1994, the patent laws were changed so that the patent term for all patents that are based in applications filed on or after June 8, 1995 is twenty years from the earliest U.S. application from which priority was claimed under 35 U.S.C. § 120. 35 U.S.C. § 154(a)(2). Second, in 1999, the patent laws were further amended to require automatic publication of applications after about eighteen months from the earliest effective filing date. 35 U.S.C. § 122(b)(1). Taken together, these two changes in the law had the effect of eliminating most potential "submarine patents" because the applications would be published and their filed open to public inspection, and preventing extension of "normal" patent term by delays in prosecution caused by applicants.

27. The Federal Circuit has only affirmed a district court or Patent Office finding of prosecution laches in two cases, and the patents in both cases were not subject to the changes in the patent laws. See *Symbol Tech*., 422 F.3d at 1386; *In re Bogese*, 303 F.3d at 1363.

28. The Federal Circuit has recognized that there may be legitimate grounds for refiling a patent application which should not normally be grounds for a holding of laches. *Symbol Tech*., 422 F.3d at 1385. As articulated by the Federal Circuit, legitimate grounds for refiling an application containing rejected claims include to present evidence of unexpected advantages of an invention when that evidence may not have been available at the time of an original rejection, or to add subject matter in order to attempt to support broader claims as the development of the invention progresses. *Id*. Even in the absence of these reasons, one may still refile as long as the refiling is not "unduly successive or repetitive." *Id*.

29. Abbot contends that, based on the totality of the circumstances, the 775 and 239 Patents are unenforceable under the doctrine of prosecution laches because they issued after unreasonable and unexcused delays. The Court has found that delays that Abbott complains about, which occurred during the prosecution of the some of the priority applications in the 775 Patent family, namely (1) the abandonment of five priority applications pre-dating February 4, 1994; (2) filing incomplete applications; (3) taking extensions of time during the prosecution of some of the priority applications; and (4) submitting incomplete drawings or sequence listings, do not amount to an unreasonable delay in prosecution.

30. Abbott also complains that Centocor's delay in presenting claims directed to human antibodies was unreasonable. The Federal Circuit has recognized that applicants are entitled to maintain pendency of an application while a competitor's product appears on the market in an effort to later draft and obtain the allowance of claims that read on the competitor's products. *State Indus., Inc. v. A.O. Smith Corp*., 751 F.2d 1226, 1235 (Fed. Cir. 1985). The Court finds that Abbott has failed to show that Centocor was aware of Abbott's development of

its human anti-TNFα antibody product, Humira, and has not met its burden of proving the Centocor unreasonably delayed the presentation of claims to human antibodies.

31. Abbott claims to have suffered prejudice in the form of economic prejudice, stemming from Centocor's alleged prosecution delays because BASF is refusing to indemnify Abbott. The Court has found that whether BASF will not indemnify Abbott for its litigation related expenses is merely speculative at this point. In any event, the Court finds that economic prejudice resulting from damages or monetary losses relating to infringement liability do not amount to the economic prejudice required to support a laches defense. *A.C. Aukerman*, 960 F.2d at 1033.

32. Abbott failed to present any evidence at trial to support it's suggestion that had it known of Centocor's human antibody claims in 2002, it could have sought a license to them during negotiations taking place between the parties. Furthermore, the argument is contradicted by Abbott's own admissions during this litigation that it thought it had an express or implied license to the 775 Patent.

33. Abbott also failed to present any evidence at trial to support its contention that it suffered from evidentiary prejudice with respect to the CDP-571 antibody because of any alleged delay. Abbott cannot meet its burden of showing prejudice in this regard.

34. Finally, to Abbott's claim that it invested billions of dollars into developing Humria and was therefore prejudiced by any alleged delay in prosecuting the 775 Patent family, this argument is simply not credible. As early as 1997, patents within the Centocor patent family began to issue, putting the public on notice that Centocor patents included, as part of the summary of the invention, human anti-TNF antibodies.

VI. INDEFINITENESS

35. Abbott has failed to show that any of the claims of the 775 and 239 Patents are invalid for indefiniteness under 35 U.S.C. § 112, ¶ 2.

36. As set forth above (Proposed Findings of Fact ¶¶ 165-170), Abbott has not shown by clear and convincing evidence that those skilled in the art would not understand the bounds of the claims or that the claims are insolubly ambiguous. Abbott has also failed to show by clear and convincing evidence that the results of competition testing will change depending on the assay conditions. The Court therefore concludes that Abbott has failed to prove that the claim term "competitively inhibits" is indefinite.

VII. CONCLUSIONS ON THE ULTIMATE ISSUES

37. This Court concludes that the 775 and 239 Patents are enforceable. Abbott has failed to demonstrate to this Court by clear and convincing evidence that Centocor committed inequitable conduct in prosecuting the 775 or 239 Patents or any of the priority applications in the 775 Patent family.

38. This Court further concludes that Abbott has failed to demonstrate to this Court that Centocor committed prosecution laches in prosecuting the 775 and 239 Patents.

39. This Court further concludes that Abbott has failed to demonstrate to this Court by clear and convincing evidence that the claim term "competitively inhibits" is invalid for indefiniteness.

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CERTIFICATE OF SERVICE

The undersigned hereby certifies that a true and correct copy of the foregoing Plaintiffs' Proposed Findings of Fact and Conclusions of Law was served via ECF filing, as follows, on September 4, 2009.

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